1: J Pharmacol Exp Ther. 1976 Mar;196(3):697-713. Related Articles, Links

Possible involvement of a transmitter different from norepinephrine in the residual responses to nerve stimulation of the cat nictitating membrane after pretreatment with reserpine.

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Pretreatment with reserpine (0.3 or 3 mg/kg, 24 hours before the experiment) reduced the norepinephrine (NE) levels in the medial muscle of the cat nictitating membrane to approximately 2% of the control values. Under these experimental conditions, the responses to postganglionic nerve stimulation were not abolished, reaching up to 50% of the maximum development of tension to exogenous sympathomimetic amines both in vivo and in vitro. In contrast to the responses to nerve stimulation obtained in normal nictitating membranes, the residual responses to nerve stimulation obtained after pretreatment with reserpine were not blocked by phentolamine (3.1 and 31 muM) or by 0.29 muM phenoxybenzamine. The effectiveness of phentolamine and phenoxybenzamine in blocking responses to exogenous NE was the same when the normal nictitating membrane was compared to the smooth muscle obtained from cats pretreated with reserpine. The residual responses to nerve stimulation were reduced when the calcium concentration in the medium was decreased to 0.65 mM. These residual responses were abolished in the presence of tetrodotoxin. Scopolamine, 0.078 muM, did not reduce the residual responses to nerve stimulation while it antagonized the responses to exogenous acetylcholine, indicating that a cholinergic mechanism is not involved in this phenomenon. Adenosine triphosphate (ATP) and adenosine diphosphosphate (ADP) behaved as agonists on the smooth muscle of the normal and of the reserpine-pretreated nictitating membrane and the responses to ATP were not blocked by phentolamine. It is concluded that the residual responses to nerve stimulation obtained after pretreatment with reserpine could be due to the release of a transmitter different from NE. The possibility that ATP or ADP might be involved in these residual responses to nerve stimulation is discussed.

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